

## LETTERS TO THE EDITOR

### Scope

Heart welcomes letters commenting on papers published in the journal in the previous six months. Topics not related to papers published earlier in the journal may be introduced as a letter: letters reporting original data may be sent for peer review.

### Presentation

Letters should be:

- not more than 600 words and six references in length
- typed in double spacing (fax copies and paper copy only)
- signed by all authors

They may contain short tables or a small figure. **Please send a copy of your letter on disk.** Full instructions to authors appear in the January 1999 issue of *Heart* (page 104).

### Non-cardiac chest pain: a useful physical sign?

EDITOR,—Non-cardiac chest pain is a major problem in general practice, in outpatients, and on the wards. Some patients are "reassured" that their pain is not cardiac but this is of course not the end of the matter. Professor Mayou and others<sup>1-3</sup> have demonstrated that these patients are still in trouble at follow up. Some are given the label "musculoskeletal" but this is not very convincing without a clear explanation. I suggest that this diagnosis can be supported in many cases by demonstrating reproduction of the pain by passive spinal movements. Production of symptoms by passive movements is well known to orthopaedic specialists.<sup>4</sup> Flexion, extension, lateral flexion, and rotation of the cervical spine, and rotation of the thoracic spine are the basic movements, and sometimes the position has to be held at the extreme for a least five seconds to reproduce symptoms—but I do not know why. This method of examination is not well known. Fifteen consecutive medical senior house officers and eight casualty officers were asked how they would demonstrate that chest pain was musculoskeletal and the best that they could do was percussion or direct pressure.

In six months of cardiology outpatients there were 27 patients (16 women and eight men) with a provisional diagnosis of angina whose history indicated another diagnosis. Their discomfort was either too unpredictable, too long or too short in duration, perhaps with paraesthesia in the fingers, or did not respond appropriately to antianginal drugs. One patient seemed to have oesophageal spasm and reflux and responded to appropriate treatment. One had pain reproduced by pressure on a costochondral joint. Of the remainder all but three had their discomfort reproduced in part or completely by passive spinal movements. Two patients also had their symptoms reproduced by percussion over the spine; both had vertebral collapse, one from osteoporosis and one from a secondary deposit. Two patients said that they were pale and sweaty during the pain, and pallor and sweating were reproduced by passive thoracic rotation and relieved by return to normal; a concurrent ECG was

unchanged. Seven patients had exercise tests, two of which were positive and they await catheterisation; their pain has not responded to antianginal medication so there is doubt that they have angina.

Of 20 consecutive patients with typical angina and positive exercise tests, only three had some discomfort in the chest on thoracic spinal rotation and they were clear that it was a different pain from their angina.

Review of the 27 patients with non-anginal pain at one year showed that seven had definite relief from non-steroidal anti-inflammatory drugs and one from physiotherapy. None had been admitted to hospital and those that still had pain seemed resigned to their symptoms but not anxious.

The exact origin of musculoskeletal pain remains obscure. It seems likely that the pain is referred along the intercostal nerves where it is exacerbated by inspiration, presumably from painful rib rotation.

Coronary disease and inflammatory spinal disease can co-exist and this physical sign does not exclude angina but its presence in the absence of any objective evidence of myocardial ischaemia—for example, no ECG changes during pain, is helpful in the differential diagnosis. Perhaps more important it demonstrates that the pain is mechanical, often allowing confident discharge without a backup nitroglycerin inhaler and giving the patient a fairly clear explanation of the symptoms, which does far more than negative tests to avoid chronic ill health.

RICHARD A BEST

Department of Cardiology, Burnley General Hospital,  
Casterton Avenue, Burnley BB10 2PQ, UK

- 1 Sanders D, Bass C, Mayou RA, *et al*. Non-cardiac chest pain: why was a brief intervention apparently ineffective? *Psychol Med* 1997;27:1033-40.
- 2 Mayou RA, Bryant BM, Sanders D, *et al*. A controlled trial of cognitive behavioural therapy for non-cardiac chest pain. *Psychol Med* 1997;27:1021-31.
- 3 Mayou R, Bryant B, Forfar C, *et al*. Non-cardiac chest pain and benign palpitations in the cardiac clinic. *Br Heart J* 1994;72:548-53.
- 4 Yates A. Spondylitis and disc lesions. In: *Mason and Curry clinical rheumatology*. London: Pitman Medical, 1980:109.

### AV node ablation and implantation of mode switching dual chamber pacemakers: effective treatment for drug refractory PAF

EDITOR,—Marshall *et al* state that the combined procedure of atrioventricular (AV) node ablation and permanent pacemaker insertion for medically refractory paroxysmal atrial fibrillation (PAF) is justified on the basis of their study results.<sup>1</sup> We agree that in many patients with this condition AV node ablation and pacemaker insertion can improve the perceived quality of life; however, we feel that it is in the patient's best interest that this procedure be performed in a staged manner with at least one month between pacemaker insertion and ablation.

Lau *et al* previously identified a group of patients with drug resistant PAF in whom DDDR pacing prevented the need for subsequent AV node ablation.<sup>2</sup> Their conclusion was that up to a third of patients with drug refractory PAF may derive benefit from sensor driven atrial pacing alone and that this treatment can result in an improvement in patient perceived quality of life, without additional AV node ablation.

Permanent blockade of the AV node results in lifelong ventricular pacemaker dependency. This can result in long term deterioration in left ventricular function,<sup>3</sup> the development of mitral regurgitation,<sup>4</sup> and symptoms of dyspnoea, tiredness, and exercise intolerance. Therefore, to proceed directly to a combined AV node ablation and pacemaker insertion procedure is to deny a significant subgroup of patients the potential benefits of not having ablation. We suggest that a trial period of rate responsive atrial pacing be undertaken in patients with drug refractory PAF before ablation. This will enable those patients who will benefit from pacing alone to be identified, and thus prevent them undergoing unnecessary AV node ablation.

We appreciate that there are potential cost implications to this staged approach; however, any increase in costs incurred may be offset by the decreased number of patients eventually requiring AV node ablation, and by the decrease in long term complications of ventricular pacing dependency.

T LEVY

S WALKER

V PAUL

Royal Brompton and Harefield NHS Trust,  
Hill End Road,  
Harefield, Middlesex UB9 6JH, UK

- 1 Marshall HJ, Harris ZI, Griffith MJ, *et al*. Atrioventricular nodal ablation and implantation of mode switching dual chamber pacemakers: effective treatment for drug refractory paroxysmal atrial fibrillation. *Heart* 1998;79:543-7.
- 2 Lau W, Paquette M, Burns, *et al*. Declining AV junction ablation for paroxysmal atrial fibrillation is associated with pacing in the atrium and improved quality of life in the PA3 trial [abstract]. *PACE* 1998;21:811.
- 3 Tse H, Lau C. Long-term effect of right ventricular pacing on myocardial perfusion and function. *J Am Coll Cardiol* 1997;29:744-9.
- 4 Le Tourneau T, Klug D, Lacroix D. Mitral valve replacement for pacing-induced severe mitral regurgitation after radiofrequency ablation of the atrioventricular node [letter]. *Heart* 1996;76:457.

This letter was shown to the authors, who reply as follows:

Levy *et al* raise an important question. The timing of AV node ablation in relation to pacing is currently the subject of some debate, although most published series describe ablation and pacing as a single procedure. However, the consensus of a recent discussion group report<sup>1</sup> was that ablation and pacing for PAF could be offered as a staged procedure to allow patients to pass the early high risk period for pacemaker lead displacement before rendering them pacemaker dependent.

There is also increasing interest in pacing alone as a mode of treatment for drug resistant PAF and this raises the suggestion that dual chamber pacing should be carried out as a standalone procedure with later ablation only if there is no improvement in symptoms. In contrast to pacing for sinus node disease (our study specifically excluded patients with significant bradycardia) the data to support pacing alone for PAF are far from clear. Levy *et al* refer to early data from the PA3 study in which some patients' symptoms improved with DDIR pacing alone.<sup>2</sup> However, data from the same study also suggested that in general DDIR pacing does not prevent atrial fibrillation and indeed shortens the time to recurrence compared to no pacing (pacemaker programmed to DDI at 30 beats/min).<sup>3</sup> Other studies of pacing alone for PAF have been disappointing. While some have shown minor reductions in atrial

fibrillation frequency,<sup>4</sup> none has demonstrated quality of life benefits. We accept that alternative atrial pacing sites and novel pacing algorithms may allow pacing to improve quality of life without the need for AV node ablation.

With regard to the complications of AV node ablation cited, the mitral regurgitation reported in two patients by le Tourneau *et al* was moderate before ablation and became severe soon after.<sup>5</sup> Improved ventricular filling (achieved by good rate control) may well worsen mitral regurgitation and we advocate that patients with moderate mitral regurgitation undergo valve surgery before ablation and pacing; in patients with PAF this may well reduce left atrial pressure sufficiently to reduce the frequency of atrial fibrillation episodes. Possible deterioration of left ventricular function associated with long term right ventricular apical pacing has to be weighed against the possibility of patients developing tachycardia induced left ventricular dysfunction if they continue to be exposed to frequent prolonged episodes of rapid atrial fibrillation; indeed, several studies have shown improvement in left ventricular systolic function after ablation and pacing for PAF.<sup>6</sup> In addition, alternative sites for right ventricular pacing (or even biventricular pacing) may reduce the potential for long term left ventricular dysfunction.

In summary, we feel that either a one or two stage procedure is acceptable. A pragmatic approach might be to offer patients the choice of ablation and pacing at one sitting, which will improve symptoms but require pacemaker dependence, or a two stage procedure, which may avoid the need for ablation but may require a second admission for symptomatic improvement. Given the high symptom burden of PAF patients being considered for ablation and pacing, we suspect many will choose the first option.

- 1 Brignole M, Gammage M, Jordaens L, *et al*. Preliminary report of a study group on ablate and pace therapy for paroxysmal atrial fibrillation [abstract]. *Arch Mal Coeur* 1998;**91**:187.
- 2 Lau W, Paquette M, Burns M, *et al*. Declining A-V junction ablation for paroxysmal atrial fibrillation is associated with pacing in the atrium and improved quality of life in the PA3 trial [abstract]. *Pacing Clin Electrophysiol* 1998;**21**:811.
- 3 Gillis A, Connolly S, Yee R, *et al*. Atrial pacing peri-ablation for paroxysmal atrial fibrillation-phase I [abstract]. *Pacing Clin Electrophysiol* 1998;**21**:872.
- 4 Murgatroyd FD, Nitzsche R, Slade AK, *et al*. A new pacing algorithm for overdrive suppression of atrial fibrillation. Chorus Multicentre Study Group. *Pacing Clin Electrophysiol* 1994;**17**(11 Pt 2):1966-73.
- 5 le Tourneau T, Lug D, Lacroix D. Mitral valve replacement for pacing induced severe mitral regurgitation after radiofrequency ablation of the atrioventricular node [letter]. *Heart* 1996;**76**:457.
- 6 Edner M, Caidahl K, Bergfeldt L, *et al*. Prospective study of left ventricular function after radiofrequency ablation of atrioventricular junction in patients with atrial fibrillation. *Br Heart J* 1995;**74**:261-7.

#### Management of polycythaemia in adults with cyanotic congenital heart disease

EDITOR,—I welcome Thorne's editorial<sup>1</sup> reiterating the pitfalls of overzealous venesection in adults with cyanotic congenital heart disease. As she states there is now a body of opinion highlighting the detrimental effects of inappropriate venesection. The evidence these conclusions are based on, however, is sparse and retrospective. This will unfortu-

nately be a feature of a relatively new field such as adult congenital disease practice until multicentred collaboration and prospective studies are planned. Despite these limitations the work we have at present points towards the principles expounded by Thorne. A recent paper by Ammash and Warnes,<sup>2</sup> not mentioned in Thorne's editorial, provides further evidence regarding the lack of association between stroke and a high haematocrit. This study of cyanotic patients followed for 3135 patient-years did not identify an association between red cell mass and stroke. Of particular interest was the finding that iron deficiency and recurrent venesection were independent risk factors for cerebrovascular events. This study not only refutes the belief that we must treat haematocrits that are "too high" but positively demonstrates the dangers of venesection. These findings endorse the previous statement from Perloff *et al* that venesection should only be performed for "temporary relief of significant, intrusive hyperviscosity symptoms".<sup>3</sup>

Unfortunately, in the UK this message is not getting through, even to cardiac specialists let alone general physicians or haematologists who may only occasionally come into contact with this patient group. In our recent survey of consultant cardiologists in Scotland,<sup>4</sup> there was a great variation in practise regarding the perceived indications for and techniques of venesection. Only a minority of consultants caring for these patients appeared to be aware of the dangers of inappropriate venesection or the appropriate management of iron deficiency. Many patients were still being submitted to regular venesection for dubious indications with no fluid replacement and no monitoring for the onset of iron deficiency. In this specialist area, presenting simple guidelines such as those drafted by Thorne to a wider audience will hopefully redress this issue and improve patient care. It would be interesting to repeat a similar national survey of consultant practise in a year or so to determine whether this message is getting through.

L SWAN  
W S HILLIS

Department of Medicine and Therapeutics,  
University of Glasgow, Western Infirmary  
Glasgow G11 6NT, UK

- 1 Thorne SA. Management of polycythaemia in adults with cyanotic congenital heart disease. *Heart* 1998;**79**:315-16.
- 2 Ammash N, Warnes CA. Cerebrovascular events in adult patients with cyanotic congenital heart disease. *J Am Coll Cardiol* 1996;**28**:768-72.
- 3 Perloff JK, Marelli AJ, Miner PD. Risk of stroke in adults with cyanotic congenital heart disease. *Circulation* 1993;**98**:1954-9.
- 4 Swan L, Birnie DH, Hillis WS. The haematological management of patients with cyanotic congenital heart disease. A time for consensus? *Eur Heart J* 1997;**18**:1973-6.

#### Myocardial infarction in young people with normal coronary arteries

EDITOR,—Williams and colleagues<sup>1</sup> recently described two patients in their early 20s presenting with acute myocardial infarction (AMI) in whom subsequent invasive investigation demonstrated no evidence of atheromatous coronary disease. In their review of the pathogenesis of AMI in patients with "normal" coronary arteries, alcohol and cocaine are discussed as potential triggers for

coronary spasm or local thrombus, but they do not mention the potential role of myocardial bridging.

Myocardial bridging is usually asymptomatic but has been related to AMI in patients as young as 15 years in the absence of risk factors for coronary artery disease and without evidence of coronary atherosclerosis.<sup>2</sup> Although a common post-mortem finding,<sup>3</sup> myocardial bridging is manifest (as segmental systolic squeezing of a coronary artery) in up to 5% of patients undergoing diagnostic angiography. As coronary arterial blood flow is primarily diastolic, the relevance of bridging in clinical practice has been the subject of extensive debate. However, recent studies suggest that deep muscle bridges can twist the coronary artery and compromise diastolic blood flow<sup>4</sup> and that this disturbance in flow at the site of the bridge might increase the propensity for intimal damage or platelet aggregation.<sup>5</sup> Intravascular ultrasound and intracoronary Doppler studies have also indicated that bridging may play an important role in AMI or angina in some patients.<sup>4</sup> Indeed, de Winter and colleagues<sup>6</sup> recently reported a case of recurrent AMI caused by a soft atheromatous plaque within a myocardial bridge. This plaque was invisible during coronary angiography and could only be imaged using intravascular ultrasound. Thus, although myocardial bridging is usually clinically irrelevant, in selected cases it can be the culprit for acute coronary syndromes. In young patients with AMI particularly, documentation of bridging during angiography may be inadequate and complete evaluation using intravascular ultrasound and Doppler is advisable.

P MUSIALEK  
A KARDOS

Department of Cardiovascular Medicine,

A P BANNING  
Department of Cardiology,  
John Radcliffe Hospital, Oxford OX3 9DU, UK

- 1 Williams MJA, Restieux NJ, Low CJS. Myocardial infarction in young people with normal coronary arteries. *Heart* 1998;**79**:191-4.
- 2 Konduracka E, Piwowarska W, Hanzlik J, *et al*. Myocardial infarction in a 15-year-old patient with a myocardial bridge narrowing the lumen of the left anterior descending artery. *Kardiologia Pol* 1994;**41**:313-16.
- 3 Ferreira AG, Trotter SE, Knig B, *et al*. Myocardial bridges: morphological and functional aspects. *Br Heart J* 1991;**66**:364-7.
- 4 Angelini P. Myocardial bridges revisited. *Cathet Cardiovasc Diagn* 1994;**32**:40-1.
- 5 Vasan RS, Bahl VK, Rajani M. Myocardial infarction associated with a myocardial bridge. *Int J Cardiol* 1989;**25**:240-1.
- 6 de Winter R, Kok WEM, Piek JJ. Coronary atherosclerosis within a myocardial bridge, not a benign condition. *Heart* 1998;**80**:91-3.

#### Value of improved treadmill exercise capacity

EDITOR,—Staniforth *et al*'s paper comparing exercise capacity in VVIR and VVI pacing modes in 12 patients with complete AV block showed that rate responsive pacing improved some but not all measures of exercise capacity.<sup>1</sup> They conclude that the best investigation for assessing exercise capacity remains unclear.

On the contrary, we feel that what is at fault is the pacing mode rather than the investigation. It is widely accepted that the preservation of AV synchrony is optimal for patients with sinus rhythm and complete AV

block and is indeed recommended by the British Pacing and Electrophysiology Group.<sup>2</sup> Maintaining AV synchrony with a physiological AV interval increases cardiac output both at rest and on exercise with normal and impaired left ventricular function.<sup>3</sup> Therefore, the use of VVIR and VVI pacing is inappropriate for six of the 12 patients in their study who were in sinus rhythm with complete AV block.

DDD (or DDDR) pacing would have been more appropriate in these patients; therefore, this study compares two sub-optimal pacing modes. Dual chamber pacing would almost certainly have resulted in better exercise capacity. The loss of AV synchrony with VVI or VVIR modes probably also accounts for the heterogeneity of the results assessing exercise capacity in this small group of patients. If dual chamber pacing had also been compared the results may well have shown an improvement in exercise capacity across the board.<sup>4</sup> The results of the UK-PACE trial comparing VVI, VVIR, and DDD modes in higher degrees of AV block are awaited,<sup>5</sup> but as with previous studies are expected to show that preservation of AV synchrony is the preferred mode on symptomatic, among other, grounds.

J D SOMAUROO  
Specialist Registrar in Cardiology,

D T CONNELLY  
Senior Lecturer in Cardiology,  
The Cardiothoracic Centre—Liverpool NHS Trust,  
Thomas Drive, Liverpool L14 3PE, UK

- 1 Staniforth AD, Andrews R, Harrison M, *et al.* Value of improved treadmill exercise capacity: lessons from a study of rate responsive pacing. *Heart* 1998;**80**:383–6.
- 2 Report of a working party of the British Pacing and Electrophysiology Group. *Br Heart J* 1991; **66**:185–91.
- 3 Kruse I, Arman K, Conradson T, *et al.* A comparison of the acute and long-term haemodynamic effects of ventricular inhibited and atrial synchronous ventricular inhibited pacing. *Circulation* 1981;**65**:846–55.
- 4 Sulke N, Chambers J, Dritsas A, *et al.* A randomized double-blind crossover comparison of four rate-responsive pacing modes. *J Am Coll Cardiol* 1991;**17**:696–706.

- 5 Toff WD, Skehan JD, De Bono DP, *et al.* The United Kingdom pacing and cardiovascular events (UK-PACE) trial. *Heart* 1997;**78**:221–3.

*This letter was shown to the authors, who reply as follows:*

DDD is the best rate response system for subjects with normal sinus node function; compared with VVIR it offers improved quality of life and less pacemaker syndrome. The evidence that the haemodynamic improvement of DDD over VVIR automatically translates into an increase in treadmill exercise capacity is not so well established,<sup>1–3</sup> and as such we do not accept the explanation of Somauroo and Connolly.

The purpose of our study was not to compare the benefits of various pacing modes, rather it was to use rate responsive pacing (in this case VVIR) as an instrument to compare the validity of various measurements of exercise capacity. It was methodologically unfortunate that some of our subjects were VVIR rather than DDD paced, but this was a reflection of the then accepted practice at the time when they had their original units implanted. The interesting observation we did make was that a treatment that is known to improve both symptoms and treadmill exercise capacity does not lead to an improvement in customary daily activity. This leaves us with the problem of divining the likely clinical benefit from the results of any study measuring treadmill exercise capacity. Just because someone can exercise harder in the laboratory does not mean that they will do so at home—but a quality of life questionnaire will show you if they do so with greater ease.

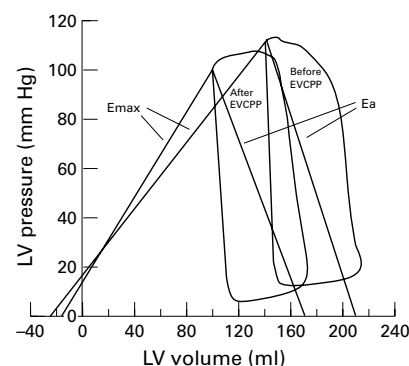
- 1 Oldroyd KG, Rae AP, Carter R, *et al.* Double blind cross over comparison of the effects of dual chamber pacing (DDD) and ventricular rate adaptive (VVIR) pacing on neuroendocrine variables, exercise performance and symptoms in complete heart block. *Br Heart J* 1991;**65**:188–93.
- 2 Linde-Edelstam C, Hjemdahl P, Pehrsson SK, *et al.* Is DDD pacing superior to VVIR? A study on sympathetic nerve activity and myocardial oxygen consumption at rest and during exercise. *PACE* 1992;**15**:425–34.

- 3 Hargreaves MR, Channon KM, Cripps TR, *et al.* Comparison of dual chamber and ventricular rate responsive pacing in patients over 75 with complete heart block. *Br Heart J* 1995;**74**:397–402.

## CORRECTION

*Effects of reconstructive surgery for left ventricular anterior aneurysm on ventriculoarterial coupling (Heart 1999;**81**:171–76).*

The incorrect Figure 5 was published in this paper. The correct figure and caption are shown below.



*Figure 5 Pressure–volume loops in a single case before and after endoventricular circular patch plasty (EVCPP) repair. Straight lines indicate the  $E_{max}$  and  $E_a$  slopes.  $E_{max}$  increased slightly (from 1.3 to 1.63 mm Hg/ml  $m^{-3}$ ), while  $E_a$  decreased (from 1.64 to 1.41 mm Hg/ml). SV did not change (from 69 to 71 ml), neither did  $V_0$  (from –16.7 to 23.3 ml). End systolic pressure decreased (from 112.85 to 100.0 mm Hg). Note the different time of maximum systolic pressure before and after surgery.*